WEST Search History

DATE: Tuesday, October 11, 2005

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L3	L2 same l1	9	L3
L2	vaccine	35885	L2
L1	CETP	265	L1

END OF SEARCH HISTORY

FILE 'H_MM' ENTERED AT [illegible]

FILE 'MEDLINE, CANCERLIT, EMBASE, BIOTECHNO, BIOSIS, WPIDS' ENTERED AT
11:48:[illegible] ON 22 OCT 2 [illegible]
L1 [illegible] S GETP AND VACCINE
L2 [illegible] DUP REM L1 (7 DUPLICATES REMOVED)
L3 [illegible] S ANTIBACTERIAL OR ANTIFUNGAL OR ANTIVIRAL
L4 [illegible] S L3 AND GETP
L5 [illegible] S MOUSE OR MURINE
L7 [illegible] S MODEL AND L4
L8 [illegible] DUP REM L4 (12 DUPLICATES REMOVED)

L[illegible] ANSWER [illegible] OF [illegible] CAPLUS COPYRIGHT [illegible] ACS

AN 199[illegible] CAPLUS

DN 12[illegible]

TI Plasmid-based **vaccine** for treating atherosclerosis

IN Thomas, Lawrence J.

PA T Cell Sciences, Inc., USA; Thomas, Lawrence J.

SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 974122[illegible]	A1	1997[illegible]	WO 1997-US7294	1997[illegible]
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2253429	AA	1997[illegible]	CA 1997-2253429	1997[illegible]
	AU 9729946	A1	19971119	AU 1997-29946	19970501
	AU 721729	B2	2000[illegible]713		
	EP 914427	A1	19990512	EP 1997-924549	19970501
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001501976	T2	[illegible]	JP 1997-539[illegible]	1997[illegible]
	US 6284533	B1	[illegible]	US 1998-171984	1998[illegible]
PRAI	US 1996-[illegible]	A	1996[illegible]		
	US 1997-[illegible]	A0	1997[illegible]		
	US 1996-[illegible]	P	1996[illegible]		
	WO 1997-US7294	W	1997[illegible]		

AB A plasmid-based **vaccine** is provided that is based on the combination of DNA segments coding for one or more B cell epitopes of **CETP** and one or more broad range helper T cell epitopes. Administration of the plasmids as a **vaccine** to a vertebrate subject provides an immune response to the subject's endogenous **CETP** and modulation of **CETP** activity, leading to prevention or reversal of various manifestations of heart disease. The vaccines provide an advantageous strategy for the prevention or treatment of atherosclerosis.

[illegible]
AU [illegible]
TI DNA plasmid based **vaccine**;
nucleic acid **vaccine** for cardiovascular disease
AU Thomas L J
PA Tadeli-Bri.
LO Needham, MA, USA.
FI WO [illegible] 7 Nov [illegible]
AI WO [illegible]-US[illegible]4 1 May [illegible]
PRAI US [illegible] 1 Feb [illegible]; US [illegible] 4 May [illegible]
DT Patent
LA English
OS WPI: [illegible]-54[illegible]1 [5]
AB A new nucleic acid **vaccine** comprises a DNA sequence I encoding an immunogenic protein, where at least 1 segment of I encodes a B-lymphocyte epitope of cholesteryl ester transferase protein **CETP** linked with at least 1 segment encoding a broad range helper T-lymphocyte epitope, where the nucleotide segment is operably linked to a promoter for directing transcription of I in a mammalian cell. Also claimed are: a DNA based plasmid **vaccine** comprising a nucleotide sequence comprising the immediate early promoter enhancer region of cytomegalo virus operably linked to a structural DNA segment encoding an immunogenic protein selected from preferred regions of a disclosed protein sequence; a DNA plasmid based **vaccine** comprising a DNA segment encoding a broad range T-lymphocyte epitope. The nucleic acid **vaccine** can be used to elevate the ratio of circulating high density lipoproteins to circulating low density lipoproteins, very low density lipoproteins or total cholesterol in a human and for reducing the level of endogenous **CETP** activity in a human. The **vaccine** can also be used to induce antibodies and for cardiovascular disease therapy. [illegible]pp

L1 ANSWER [illegible] EMBASE [illegible]
AN [illegible] EMBASE
TI Genetic polymorphisms and activity of cholesteryl ester transfer protein **CETP** : Should we be measuring them?
AU Ordovas J.M.
CS J.M. Ordovas, Lipid Metabolism Laboratory, Jean Mayer USDA Hum. Nutr. Res. Ctr., Tufts University, Boston, MA, United States. Ordovas@hnrc.tufts.edu
SO Clinical Chemistry and Laboratory Medicine, [illegible]
Vol: [illegible]
ISSN: [illegible] ISBN: [illegible]
CY Germany
DT Journal; Article
FS [illegible] Cardiovascular Diseases and Cardiovascular Surgery
[illegible] Human Genetics
[illegible] Clinical Biochemistry
[illegible] Drug Literature Index
LA English
SL English
AB Cholesteryl ester transfer protein **CETP** is a plasma glycoprotein that mediates the transfer of cholesteryl ester from high density lipoproteins HDL to triglyceride-rich lipoproteins in exchange for triglycerides. Several approaches are currently being used in research laboratories to measure its activity and or mass. However, these assays are not standardized and it is not possible to compare data from different laboratories. Also, we lack enough information to assess the value of this variable as a coronary heart disease CHD predictor. Several genetic variants at **CETP** locus have been identified and they have been generally associated with increased HDL- cholesterol concentrations. However, there is no consensus about the association of this **CETP** -related increase in HDL-cholesterol and protection against CHD. Nevertheless, the most recent evidence from the common **CETP** -TaqI-B polymorphism shows that the lower **CETP** activity associated with the presence of this polymorphism decreases CHD risk in men. Based on this and previous evidence, there has been an interest in the development of **CETP** inhibitors as a tool to increase HDL-cholesterol, thus reducing CHD risk. However, it should be noted that the evidence about the cardioprotective role of these drugs is not yet available.

L? ANSWER 4 OF 1? MEDLINE [illegible]
AN [illegible] MEDLINE
DN [illegible] PubMed ID: [illegible]
TI **Vaccine**-induced antibodies inhibit **CETP** activity in vivo and reduce aortic lesions in a rabbit model of atherosclerosis.
CM Comment in: Arterioscler Thromb Vasc Biol. [illegible]
AU Rittershaus C W; Miller D P; Thomas L J; Picard M D; Honan C M; Emmett C D; Pettey C L; Adari H; Hammond R A; Beattie D T; Callow A D; Marsh H C; Ryan U S
CS AVANT Immunotherapeutics, Inc., Needham, MA [illegible], USA.. crittershaus@avantimmune.com
NC HL-[illegible] NHLBI
SO ARTERIOSCLEROSIS, THROMBOSIS, AND VASCULAR BIOLOGY, [illegible] Sep [illegible] 2106-12.
Journal code: 95[illegible]. ISSN: 1524-4636.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM [illegible]
ED Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]
AB Using a **vaccine** approach, we immunized New Zealand White rabbits with a peptide containing a region of cholesteryl ester transfer protein (**CETP**) known to be required for neutral lipid transfer function. These rabbits had significantly reduced plasma **CETP** activity and an altered lipoprotein profile. In a cholesterol-fed rabbit model of atherosclerosis, the fraction of plasma cholesterol in HDL was 42% higher and the fraction of plasma cholesterol in LDL was 24% lower in the **CETP**-vaccinated group than in the control-vaccinated group. Moreover, the percentage of the aorta surface exhibiting atherosclerotic lesion was 39.6% smaller in the **CETP**-vaccinated rabbits than in controls. The data reported here demonstrate that **CETP** activity can be reduced in vivo by vaccination with a peptide derived from **CETP** and support the concept that inhibition of **CETP** activity in vivo can be antiatherogenic. In addition, these studies suggest that vaccination against a self-antigen is a viable therapeutic strategy for disease management.

L1 ANSWER [illegible]
AN [illegible]
DN [illegible]
TI An immunotherapeutic approach for the treatment of low plasma HDL-cholesterol
AU Ryan, Una S.; Fitzsimmons, Charles W.
CS AVANT Immunotherapeutics, Inc., Needham, MA, [illegible], USA
SO NATO Science Series, Series I: Life and Behavioural Sciences [illegible], Vascular Endothelium, [illegible]
CODEN: [illegible]; ISBN: [illegible]
PB IOS Press
DT Journal
LA English
AB One determinant of plasma HDL-cholesterol concn. is cholesteryl ester transfer protein CETP activity. Inhibition of CETP activity increases plasma HDL-C, thus providing a potential therapeutic target for the treatment of atherosclerosis. Using a vaccine approach, we immunized New Zealand White rabbits with a peptide contg. a region of CETP known to be required for neutral lipid transfer function. CETP-vaccinated rabbits had significantly reduced plasma CETP activity and an altered lipoprotein profile compared with control rabbits. In a cholesterol-fed rabbit model of atherosclerosis, the fraction of plasma cholesterol in HDL was 42% higher, and the fraction of plasma cholesterol in LDL was 24% lower in the CETP-vaccinated group compared with the control-vaccinated group. Moreover, the percentage of the aorta surface exhibiting atherosclerotic lesion was 39.6% smaller in the CETP-vaccinated rabbits compared with controls. The data reported here demonstrate that CETP activity can be reduced in vivo by vaccination with a peptide derived from CETP, and support the concept that inhibition of CETP activity in vivo can be anti-atherogenic. Currently, this vaccine is in clin. trials.
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVA

[illegible]

AN [illegible] BIOSIS

DN [illegible]

TI Plasmid-based **vaccine** for treating atherosclerosis.

AU Thomas, Lawrence [illegible]

CS [illegible] Easton, MA USA

ASSIGNEE: AVANT Immunotherapeutics, Inc.

PI US [illegible] September 4, [illegible]

SO Official Gazette of the United States Patent and Trademark Office Patents, Sep. 4, [illegible] Vol. [illegible], No. 1, pp. No Pagination. e-file.
ISSN: [illegible]

DT Patent

LA English

AB A plasmid-based **vaccine** is provided herein based on the combination of DNA segments coding for one or more B cell epitopes of cholesteryl ester transfer protein **CETP** and one or more broad range helper T cell epitopes. Administration of the plasmids as a **vaccine** to a vertebrate subject provides an immune response to the subject's endogenous **CETP** and modulation of **CETP** activity, leading to prevention or reversal of various manifestations of heart disease. The vaccines provide an advantageous strategy for the prevention or treatment of atherosclerosis.

L? ANSWER [illegible] BIOTECHNO [illegible] COPYRIGHT [illegible] THOMSON DERWENT AND ISI
AN [illegible] BIOTECHNO
TI Cholesteryl ester transfer protein corrects dysfunctional high density lipoproteins and reduces aortic atherosclerosis in lecithin cholesterol acyltransferase transgenic mice;
adeno virus vector-mediated human phosphatidylcholine-sterol-O-acyltransferase expression in mouse for **artherosclerosis model**

AU Foger B; Chase M; Amar M J; Vaisman B L; Shamburek R D; Paigen B; Fruchart-Najib J; Paiz J A; Koch C A; Hoyt R F; Brewer Jr H B; *Santamarina-Fojo S
CS Nat.Inst.Health-Bethesda; Jackson-Lab.Bar-Harbor; INSERM
LO National Institutes of Health, Molecular Disease Branch, NHLBI, Building 10, Room 7N115, 10 Center Drive MSC 1666, Bethesda, MD 20892-1666, USA.
SO J.Biol.Chem.; (1999) 274, 52, 36912-20
CODEN: JBCHA3 ISSN: 0021-9258
DT Journal
LA English
AB Human phosphatidylcholine-sterol-O-acyltransferase (PSCA, EC-2.3.1.43) expression (adeno virus vector) in mice (PSCA-Tg) leads to increased high density lipoprotein (HDL) cholesterol levels but paradoxically, enhanced atherosclerosis. PSCA-Tg were cross bred with cholesteryl ester transfer protein (**CETP**)-Tg mice to test the hypothesis that the absence of **CETP** in PSCA-Tg mice facilitates the accumulation of dysfunctional HDL leading to impaired reverse cholesterol transport and the development of a pro-atherogenic state. Expression if **CETP** in PSCA-Tg mice reduced total cholesterol, reflecting a decrease in HDL cholesterol levels. **CETP** normalized both the plasma clearance of (3H cholesteryl esters (3H CE) from HDL as well as the liver uptake of (3H CE from HDL in PSCA-Tg mice. **CETP** expression reduces atherosclerosis in PSCA-Tg mice by restoring the functional properties of PSCA-Tg mouse HDL and promoting the hepatic uptake of HDL-cholesteryl ester. Therefore **CETP** expression is beneficial in pro-atherogenic states that result from impaired reverse cholesterol.
(64 ref)

AN [illegible] EMBASE [illegible]
AN [illegible] EMBASE
TI Molecular mechanisms, lipoprotein abnormalities and atherogenicity of hyperalphalipoproteinemia.
AU Yamashita S.; Maruyama T.; Hirano K.; Sakai N.; Nakajima N.; Matsuzawa Y.
CS S. Yamashita, Department of Internal Medicine, Graduate School of Medicine, Osaka University, 1-1 Yamadaoka, Suita, Osaka 565-0871, Japan. shirus@sb.med.osaka-u.ac.jp
SO Atherosclerosis, [illegible]
Refs: [illegible]
ISSN: [illegible] CODEN: ATHSBL
PUI [illegible]
CY Ireland
DT Journal; General Review
FS 017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
025 Hematology
029 Clinical Biochemistry
036 General Pathology and Pathological Anatomy
LA English
SL English
AB Hyperalphalipoproteinemia (HALP) is caused by a variety of genetic and environmental factors. Among these, plasma cholesteryl ester transfer protein (**CETP**) deficiency is the most important and frequent cause of HALP in the Asian populations. **CETP** facilitates the transfer of cholesteryl ester (CE) from high density lipoprotein (HDL) to apolipoprotein (apo) B-containing lipoproteins, and is a key protein in the reverse cholesterol transport system. The deficiency of **CETP** causes various abnormalities in the concentration, composition, and function of both HDL and low density lipoprotein (LDL). The significance of **CETP** in terms of atherosclerosis had been controversial. However, the in vitro evidence showed large CE-rich HDL particles in **CETP** deficiency are defective in cholesterol efflux. Similarly, scavenger receptor BI (SR-BI) knockout mice show a marked increase in HDL-cholesterol but accelerated atherosclerosis in atherosclerosis-susceptible mice. Recent epidemiological studies in Japanese-Americans and in Omagari area where HALP subjects with the intron 14 splicing defect of **CETP** gene are markedly frequent, have demonstrated an increased incidence of **coronary** atherosclerosis in **CETP** deficient patients. Thus, **CETP** deficiency is a state of impaired reverse cholesterol transport which may possibly lead to the development of atherosclerosis. The current review will focus on the molecular mechanisms and atherogenicity of HALP, especially **CETP** deficiency.

AN [illegible] EMBASE [illegible]
AN [illegible] EMBASE
TI Cholesteryl ester transfer protein inhibitors.
AU Shinkai H.
CS H. Shinkai, Central Pharmaceutical Res. Inst., JT Inc., 1-1 Murasaki-cho, Takatsuki, Osaka 569-1125, Japan. hisashi.shinkai@jtis.jti.co.jp
SO Expert Opinion on Therapeutic Patents, [illegible]. 11 [illegible]-745.
Vol: 11
ISSN: 1354-3776. CODEN: EOTPEG
CY United Kingdom
DT Journal; General Review
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LA English
SL English
AB As well as hypercholesterolaemia, low levels of high-density lipoprotein cholesterol (HDL-C) are critical risk factors for atherosclerosis and coronary heart disease (CHD). Although fibrate, simvastatin and niacin can be used for the treatment of a low HDL-C level, their efforts, however, are not wholly satisfactory. Thus, better drugs for the elevation of HDL-C are desired. Among the many methods that may be used to raise HDL-C levels, this review focuses on small molecule inhibitors of cholesteryl ester transfer protein (CETP) and summarises recent patent and journal data.